Alcohol-Related Metabolic Emergencies

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KEYWORDS

• Alcohol • Wernicke • Alcohol use disorder • Alcoholic Ketoacidosis
• Encephalopathy

KEY POINTS

- In your intoxicated patients, do not just assume they are simply intoxicated by alcohol. Evaluate for hypoglycemia, lactic acidosis, and hyponatremia.
- Suspect alcoholic ketoacidosis in patients who have been on an alcohol binge without eating with nausea and vomiting. Do not forget the venous blood gas and initiate treatment with dextrose-containing fluids.
- Wernicke encephalopathy (WE) is reversible and only one-third of WE patients will have the triad of altered mental status, oculomotor dysfunction, and gait ataxia.
- Korsakoff syndrome is irreversible so do not delay high dose IV thiamine administration during the presumed diagnosis of WE.

INTRODUCTION

Alcohol-related disorders are a common and often life-threatening cause of emergency department (ED) presentations. Alcohol use disorder (AUD) in the United States has an increased prevalence in ED visits over previous years.[1] Most of the patients with AUD that are at highest risk for ED visits or hospitalizations had higher incidence of homelessness, concomitant depression, and injury or trauma.[1] Therefore, these patients who present to the ED with alcohol intoxication are some of the higher risk patients seen in the ED. Alcohol has known effects on several organ systems that are widely known such as the central nervous system and gastrointestinal system in chronic users. However, alcohol also has effects on the endocrine and metabolic system that can result in significant complications if unrecognized by emergency clinicians caring for these patients. Successful management requires an understanding of the effects of alcohol on the metabolic and endocrine systems. This is a review

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of the most common alcohol-related metabolic emergencies including alcoholic ketoacidosis (AKA), Wernicke–Korsakoff, lactic acidosis, hypoglycemia, and hyponatremia.

ALCOHOLIC KETOACIDOSIS

AKA is a metabolic disorder classified by alcohol use along with the presence of ketoacidosis. It is a common cause of ketoacidosis in nondiabetic patients and is very likely underdiagnosed. This underdiagnosis is most likely due to the rapid improvement with resolution of vomiting with antiemetics and subsequent nourishment with food. Therefore, emergency clinicians likely do not obtain laboratories and make this diagnosis. So, the true incidence of AKA is unknown. According to one study, risk factors for development of AKA include lower body mass index (BMI), whiskey drinkers, smokers, and baseline hypoglycemia.[2] The mortality for AKA is likely also underreported due to the assumption that patients with chronic alcohol use have likely died of the more common etiologies such as alcoholism or another alcohol-related disease (ie, cirrhosis, trauma).

Pathophysiology

The pathophysiology of AKA depends on several conditions to be present. There needs to be malnutrition, increased nicotinamide adenine dinucleotide hydrogen (NADH), and hypovolemia.[3] Owing to the malnutrition, the body enters a starvation state that leads to the production and release of glucagon to provide the body with some nourishment, primarily glucose from gluconeogenesis and glycogenolysis. However, this takes a bit of time to create glucose, so the body begins lipolysis. Lipolysis leads to an increased synthesis of beta-hydroxybutyrate and other serum ketone bodies resulting in metabolic acidosis and subsequent symptomology. During a period of decreased alcohol consumption, caloric intake falls, and lipolysis increases. This creates a cycle of increasing cortisol, norepinephrine, and glucagon levels. As a result, an increased number of fatty acids are released from peripheral triglycerides and undergo beta-oxidation to generate acetyl-CoA. The capacity of the Krebs cycle is exceeded, and oxidation must be achieved through ketogenesis.[4–6] In the presence of alcohol, pyruvate is preferentially converted to lactic acid leading to increased acidosis. The increased NADH does not allow gluconeogenesis to occur due to decreased pyruvate which contributes to the euglycemia or hypoglycemia seen in AKA.[7] Last, alcohol also induces volume depletion. Alcohol is an antagonist to free water reabsorption by inhibiting antidiuretic hormone as well as the increased risk for vomiting which can occur with AKA. Because of the reduced volume intravascularly, there is decreased clearance of ketones in the urine leading to increase ketone bodies in the serum and subsequent ketoacidosis.[8]

Clinical Presentation

The typical patient with AKA usually has AUD, poor nutrition, and presents in a varying degree of alcohol withdrawal after cessation. Owing to their chronic malnourishment, they have a significant decrease in caloric intake and commonly exhibit symptoms of nausea, vomiting, abdominal pain, and dehydration.[9–11] Because of the degree of acidosis present, they will commonly present with tachypnea due to the body's respiratory compensation for the metabolic acidosis. Other vital sign abnormalities include tachycardia and hypotension, but they may present with normal or elevated blood pressures due to alcohol withdrawal. The presentation may include complications such as pancreatitis, gastritis, or esophagitis with or without gastrointestinal bleed,

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which may be caused by the AKA or may precipitate the AKA.[12] Because plasma osmolality remains grossly normal, they do not have marked altered mental status (AMS) until their acidosis is profound.[9,11] In addition, because of their AUD, emergency clinicians should evaluate for alcohol withdrawal, Wernicke encephalopathy (WE), and other common complications associated with chronic alcohol use.

Workup and Differential Diagnosis

Laboratory evaluations should include but not be limited to venous blood gas (VBG), basic metabolic panel (BMP), liver function tests, magnesium, prothrombin time (PT), partial thromboplastin time (PTT), lactic acid level, lipase, serum ketones, urine ketones, and beta-hydroxybutyrate. Serum and urine ketone assays rely on nitroprusside testing which detects the presence of acetoacetate and does not detect beta-hydroxybutyrate. Therefore, do not rely on negative serum or urine ketone testing. If serum beta-hydroxybutyrate testing is available, it should be used. The presence of an elevated anion gap without acidosis should alert the emergency clinician that there may be a mixed acid–base disorder present. Almost universally, the anion gap will be elevated in AKA, but if there is a mixed acid–base disorder present, the serum bicarbonate and/or the pH on a VBG may be close to normal. Lactic acid levels in AKA are commonly elevated but not severely (>4 mmol/L). If the lactic acid level is severely elevated, another concomitant condition may be present and should be investigated for such as sepsis, liver dysfunction, or seizures.[8] The diagnostic criteria for AKA can be found in **Box 1**.

Additional studies that should be obtained include an electrocardiogram (EKG) and imaging for any other concomitant disease process that may be occurring. Although abdominal tenderness in AKA is common, it should not be peritoneal so the presence of any signs of a surgical abdomen should warrant prompt evaluation.[3] An EKG can quickly evaluate for signs of acute coronary syndrome; however, more importantly, it can evaluate for any concomitant hyperkalemia that may develop due to acidemia. Despite an elevated serum potassium measurement, total body potassium levels are low and are falsely elevated due to the acidemia. If concomitant hyperkalemia is present with EKG changes, prompt treatment with calcium is warranted; however, further treatment with potassium elimination is usually not recommended.

The differential diagnosis for a patient presenting with AKA is expansive. Other conditions to consider in an alcoholic patient with abdominal pain, nausea and vomiting should include but not be limited to gastritis, esophagitis, peptic ulcer disease, and pancreatitis. For the patient presenting with signs of elevated anion gap acidosis, the included conditions should also be diabetic ketoacidosis, lactic acidosis, and toxic alcohol intoxication. DKA is distinguished from AKA by an elevated serum glucose of greater than 250. Both DKA and fasting or euglycemic ketoacidosis are distinguished

Box 1
Diagnostic criteria for alcoholic ketoacidosis

- History of alcoholic use disorder or alcohol dependence
- Euglycemia or hypoglycemia
- Anion gap metabolic acidosis
- Presence of urine or serum ketones (beta-hydroxybutyrate is the predominant ketone present)
- Mildly elevated (approximately 20 mm/kg) osmolal gap

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from AKA by a normal osmolal gap, which is usually elevated in AKA.[8] In addition, toxic alcohols are distinguished from AKA by the absence of ketones, which are usually elevated in AKA.

Management

Prompt recognition and treatment of AKA are what has prevented an elevated mortality. The mainstay of treatment for AKA consists of intravenous (IV) fluid resuscitation and glucose and electrolyte replenishment. The goal of treatment is to replenish volume that has been lost through vomiting and diuresis as well as to slow the rate of ketogenesis and allow the metabolization and urinary clearance of ketone bodies.[9,12,13] Balanced electrolyte solutions are the preferred choice for the initial stages of IV resuscitation, as normal saline solution may worsen a hyperchloremic non-anion gap metabolic acidosis. For patients with impaired gluconeogenesis, a dextrose (D5/D10) drip is recommended.[3,8,9,11,13,14] Dextrose infusion running concomitantly with the isotonic fluid will increase insulin secretion and decrease glucagon secretion which will slow down ketogenesis and increase the body's metabolization of present ketone bodies by providing a better energy substrate for the brain. Dextrose 5% as a mixture with the isotonic fluid bag or running concomitantly is usually sufficient in euglycemic patients in AKA. Hypoglycemic patients should be treated aggressively with higher concentrations of dextrose such as D10 or D50. Patients who are subsequently found to have elevated blood glucose may require an insulin infusion with a dextrose infusion running concomitantly.

Electrolytes, particularly potassium, should be replenished intravenously as dictated by the patient's laboratory analysis.[3,8,14–17] As stated previously, patients with AKA often have falsely elevated potassium levels despite having total body potassium stores that are diminished. Before the administration of dextrose (or insulin), potassium levels should be checked, and hypokalemia should be corrected before administration as dextrose or insulin infusions are likely to shift the potassium into the cells and subsequently lower serum potassium levels. Potassium chloride either orally, if tolerant, or IV is recommended. Be mindful of the serum magnesium level as well. Often, alcoholic patients are magnesium-depleted as well, and concomitant hypokalemia often occurs. Therefore, magnesium repletion in patients who are hypomagnesemic is required to help replenish potassium. Lastly, 500 mg of IV thiamine should be administered in patients at risk of WE, which will be discussed later.[18,19] Most patients with AKA who are able to tolerate orally, have resolution of their ketoacidosis, and electrolytes repleted can be safely discharged home. All others should be admitted for further management.

WERNICKE–KORSAKOFF

Wernicke–Korsakoff syndrome is a bimodal disease process that results from thiamine (vitamin B1) deficiency. Although it is present in several conditions, including malnutrition, AIDS, and hyperemesis gravidarum, it is most commonly seen in patients with AUD. WE is the acute presentation of thiamine deficiency and is reversible if treated promptly, whereas Korsakoff syndrome (KS) is the irreversible neurologic condition that results from chronic thiamine deficiency.[20] The prevalence and incidence of these disorders is very underreported and underestimated. It is commonly missed in the clinical setting with adults being missed 75% to 80% of the time and in children 58% of the time.[21] Misdiagnosis has higher chance when alcohol-intoxicated patients present to the ED.[21,22] Most studies report a prevalence as high as 2.8% worldwide with some diagnosed on autopsy.[21] The incidence has been decreasing due to

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developed countries supplementing foods with vitamins including thiamine. Therefore, a higher incidence occurs in developing countries that are unable to fortify their foods with vitamins. The estimated mortality is around 17% and 80% of patients who develop WE go on to develop KS.[21] It is more common in men than women and can also occur in children.

Pathophysiology

Thiamine is a necessary substrate for the brain. Thiamine is absorbed in the gastointestinal (GI) tract and is used to create thiamine pyrophosphate, which is necessary for carbohydrate metabolism and creation of adenosine triphosphate (ATP), lipid metabolism for creation of myelin sheaths, and production of amino acids and glucose-derived neurotransmitters.[21] Patients with AUD have thiamine deficiency from decreased caloric intake, reduced absorption, decreased hepatic storage, and poor utilization.[23] When thiamine is deficient, neuronal injury results from inhibited metabolism in areas of the brain with high thiamine turnover. As this injury occurs, glucose utilization is impaired causing further neuronal injury, increase in reactive oxygen species, and increased N-methyl-D-aspartate (NMDA) receptor-mediated excitotoxicity, causing the blood–brain barrier to break down resulting in petechial hemorrhages, microglial proliferation, and vascular congestion.[19,21,24,25] With frequent WE episodes, demyelination and neuronal loss occurs, particularly in the thalamus and mammillary bodies.[26] Chronic demyelination and neuronal death lead to chronic lesions, which leads to KS.

Clinical Presentation

WE classically presents with the triad of altered mental status (AMS), oculomotor dysfunction, and/or gait ataxia. However, less than 33% of patients will present with all three findings with 19% of patients presenting with none.[20,21,27] The spectrum of thiamine deficiency can vary the symptomology greatly. Patients with acute thiamine deficiency often lead to encephalopathy, whereas chronic long-term deficiency leads to peripheral polyneuropathy. Patients with acute deficiency present with AMS due to encephalopathy, which presents as disorientation and indifference. Oculomotor dysfunction is caused by lesions within the visual pathway nuclei and presents as conjugate gaze palsies, lateral rectus palsy, and nystagmus. Patients can also manifest anisocoria and pupillary sluggishness.[28] Gait ataxia is due to vestibular dysfunction, cerebellar involvement, and polyneuropathy and presents as gait abnormalities, particularly during tandem gait evaluation. The difficulty arises in that all these signs and symptoms are impossible to differentiate from alcohol intoxication.

KS is a chronic disturbance in memory and cognition and presents with selective retrograde or anterograde amnesia without change in orientation. Patients are often awake and alert but have difficulty with memory and learning. There are no accepted diagnostic criteria for KS as the clinical presentation is extremely variable.[29] Confabulation is sometimes seen. Many patients will have a history of subclinical rather than overt WE.[19,30–32]

Workup and Differential Diagnosis

WE and KS are clinical diagnoses, which, as stated before, a difficult one to make. Therefore, the emergency clinician must maintain a high level of suspicion for these diagnoses. The Caine criteria may aid in diagnosis of WE, whereas the classic triad may miss two-thirds of cases. The Caine criteria have 85% sensitivity for diagnosing WE when two of the criteria are met. The Caine criteria are shown in **Box 2**.

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Laboratory and radiographic analysis are primarily used to rule out other serious alternative diagnoses. Laboratory evaluations should include but not be limited to complete blood count (CBC), BMP, hepatic panel, ammonia, PT, PTT, and magnesium. Serum thiamine levels need not be measured, as treatment should be initiated before results are available.[33] In addition, serum thiamine levels have poor sensitivity and specificity for true thiamine levels. Thiamine is present more in the serum as thiamine pyrophosphate and the results of this test are not readily available in the ED setting. A non-contrast computerized tomography (CT) head should be ordered in these patients to evaluate for cerebral edema, herniation due to hyperammonemia, and alternative causes of AMS such as traumatic intracerebral hemorrhage or tumor. CT of the head is poorly sensitive and specific for WE and can only be visualized non-emergently on MRI.[34–36]

Differential diagnosis should include but not be limited to alcohol-induced psychosis, AKA, hepatic encephalopathy, delirium tremens, structural brain lesions, infection, toxidrome, trauma, and other metabolic or endocrine causes. In addition, some of these conditions may occur concomitantly with WE, so WE should not be ruled out based on the presence of these other disease processes.

Management

Once the diagnosis of WE is considered, thiamine should be administered. The recommended dose is 500 mg of IV thiamine, ideally before the administration of glucose. Little evidence supports the suspicion that giving glucose before thiamine will worsen WE.[18,19] Patients who are hypoglycemic should be given glucose to prevent the effects of hypoglycemia and then administered thiamine. Patients who have prolonged supplementation without the administration of thiamine are at higher risk for developing WE.[19] Clinical improvement may occur within a day of thiamine administration and begins with oculomotor improvement. Gait ataxia and vestibular dysfunction are last to correct and may require 2 weeks of treatment.[19,37] Most patients with WE require admission to the hospital for further thiamine supplementation.

KS is considered a permanent neurologic disorder, and most patients with diagnosed KS require social support or a long-term care facility.[20,30,38]

ALCOHOL USE DISORDER-RELATED METABOLIC DISTURBANCES

Although many electrolyte abnormalities and metabolic disturbances can be seen in patients with AUD, hypoglycemia and hyponatremia are the most urgent.

Hypoglycemia

As described earlier, habitual alcohol users are at an increased risk of hypoglycemia. However, the true incidence is unknown as several studies have shown that blood glucose levels measured in adult patients with alcohol intoxication are rarely

Box 2
The Caine criteria for the diagnosis of Wernicke encephalopathy[26]

Must have two of the following criteria:
1. Dietary deficiencies
2. Oculomotor abnormalities
3. Cerebellar dysfunction
4. Either an acute or subacute AMS or mild memory impairment.

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low.[39,40] Despite these studies, fingerstick glucose should still be checked in intoxicated patients so as to not miss hypoglycemia.

Pathophysiology

Alcohol ingestion causes mechanisms that increase the risk of hypoglycemia. In acute ingestion, the metabolization of ethanol into acetic acid causes an increase in NADH, which leads to an increase in the conversion of pyruvate to lactic acid and impairment of gluconeogenesis. In chronic ingestion, hepatocytes have decreased levels of glycogen stores to contribute to glycogenolysis, predisposing the body to rely on gluconeogenesis which is already impaired. Decreased levels of biotin and thiamine, required cofactors for pyruvate metabolism, causes pyruvate to be converted to lactic acid rather than acetyl coenzyme A (acetyl-CoA). In patients with cirrhosis, lactate clearance by the liver is also reduced leading to increased levels, which further exacerbates the above events.[41] Chronic users are also malnourished and substitute alcohol intake for dietary intake. These effects are exaggerated in children with even small amounts of alcohol and these patients are at much higher risk of developing hypoglycemia with alcohol intake.

Clinical Presentation

The presentation of hypoglycemia ranges from subclinical to seizures, coma, and death. The classic presentation is referred to as Whipple's triad, which is clinical signs of hypoglycemia, blood glucose that is low, and resolution of those symptoms when glucose is normalized. Hypoglycemia signs and symptoms are broken down into autonomic and neuroglycopenic symptoms. Autonomic is the body's attempt to mobilize glucose and is caused primarily by catecholamines and includes tachycardia, hypertension, diaphoresis, and agitation. Shortly, if not corrected, the neuroglycopenic symptoms begin which is caused by neuronal injury and includes confusion, seizures, obtundation, and death, if prolonged.

Workup

The evaluation of an altered patient should always include a point-of-care glucose level. The early recognition of hypoglycemia leads to better outcomes. It is often missed in the acutely intoxicated patient as the assumption remains that the patient is simply intoxicated. Further laboratory and radiographic analysis are mostly to rule out other potential causes of altered sensorium and should include but not be limited to serum lactate, CBC, BMP, hepatic panel, VBG, C-reactive protein, PTT, PT, serum and urine ketones, and serum osmolality.

Lactic acid is often elevated in patients with alcohol intoxication and can occur concomitantly with hypoglycemia. Lactic acid levels of greater than 5 mmol/L with a pH of less than 7.35 should suggest an etiology other than alcohol-related lactic acidosis.[41,42]

Management

Volume resuscitation with IV fluids with dextrose is the mainstay of management of patients with hypoglycemia. Oral supplementation is best but IV should be initiated in patients with inability to tolerate orally.

Hyponatremia

Pathophysiology

Hyponatremia is defined as a sodium level of less than 135 mmol/L. Severe hyponatremia occurs at less than 125 mmol/L. In the acute alcohol-intoxicated patient,

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hyponatremia is typically hypovolemic and due to excessive diuresis and GI fluid losses. Reversible tubular injury leading to increased solute excretion may also contribute.[43]

In patients with chronic AUD and cirrhosis, hypervolemic hyponatremia can occur and is due to an inability to excrete enough solute-free water. This is the result of both decreased circulating volumes causing an increase in vasopressin and of a decreased glomerular filtration rate (GFR) causing sodium imbalances in the renal tubules.[44] Alternatively, hyponatremia may be euvolemic and due to beer potomania. Beer potomania occurs when urinary dilution is intact, but a large fluid intake and a decreased dietary solute intake, particularly protein, lead to low solute excretion and a diminished excretion of electrolyte-free water.[45] Last, patients with AUD can have pseudohyponatremia due to alcohol-induced hypertriglyceridemia and subsequent pancreatitis.[43]

Clinical Presentation

The presentation of hyponatremia ranges from mild generalized weakness and nausea/vomiting to AMS and seizure. Owing to the multiple different AUDs that present with similar signs and symptoms, hyponatremia from alcohol use is often misdiagnosed. Alcohol withdrawal, acute alcohol intoxication, WE, and hypoglycemia can all present very similar to hyponatremia and should be considered on the differential. Seizures and coma occur due to cerebral edema that results from an acute decrease in sodium, typically within less than 48 hours.[46]

Workup

Laboratory evaluation should be obtained in all patients with altered sensorium with no visible explanation or prolonged obtundation. The pitfall comes when the acutely intoxicated patient presents, and the assumption is simple intoxication and other more serious conditions are missed. Laboratory studies should include but not be limited to serum lactate, CBC, BMP, and hepatic panel. An increased serum urea/creatinine ratio and a low urine sodium concentration indicate hypovolemic hyponatremia.[43] Serum osmolality can be measured but in acute intoxication may be normal. A reduction in plasma osmolality may be offset by ethanol because ethanol can freely cross cell membranes. A head CT should be considered in all patients and is recommended in any patients with trauma.

Differential diagnosis should include but not be limited to other causes of hypovolemic hyponatremia such as dehydration and diuretic use, other causes of euvolemic hyponatremia such as Syndrome of Inappropriate Antidiuretic Hormone (SIADH), primary polydipsia, and hypothyroidism, and other causes of hypervolemic hyponatremia such as congestive heart failure and kidney disease. Other electrolyte abnormalities and other causes of cerebral edema should also be considered.

Management

The spectrum of the different presentations really helps with deciding acute and aggressive treatment versus slow and conservative treatment. In alcohol-related hyponatremia, which is usually acute, serum sodium should be corrected by less than 10 mmol/L in the first 24 hours and less than 18 mmol/L in the first 48 hours. If asymptomatic, this may be done with simple fluid restriction, oral salt repletion, or IV fluids resuscitation with salt-containing fluids. Although normal saline is often appropriate, severe hyponatremia, characterized by the presence of seizure or coma, should be treated with 3% saline. Sodium levels should be trended every

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2 hours, and if overcorrected, a D5W infusion can be started and titrated to urine output.[47,48]

SUMMARY

Alcohol-related metabolic emergencies are commonly encountered in the ED. Appropriate management relies on rapid identification and an understanding of each disorder's pathophysiology. Although most disorders will require additional inpatient management, initial resuscitation must be initiated in the ED.

CLINICS CARE POINTS

Pearls
- Remember the diagnosis of alcoholic ketoacidosis. Fluid resuscitate with isotonic balanced solutions and provide dextrose supplementation. Consider insulin in your hyperglycemic patients concomitantly with the dextrose infusion. Replenish electrolytes, especially potassium and magnesium.
- Wernicke encephalopathy (WE) is reversible. Consider this diagnosis which is difficult to distinguish from alcohol intoxication. The Caine criteria can help make the diagnosis, whereas the classic triad can miss the diagnosis. Give high-dose thiamine 500 mg IV without delay.
- Electrolyte disturbances are common, but the emergency clinician should not assume simple intoxication. Evaluate for these disturbances. Treat acute, severe hyponatremia with 3% saline—correct by less than 10 mmol/L in the first 24 hours.

Pitfalls
- Alcoholic ketoacidosis: Delaying dextrose-containing fluids that may lead to hypoglycemia and worsen the metabolic acidosis.
- WE: Missing this diagnosis by relying on the lack of the classic triad. Delaying treatment while waiting for a serum thiamine level, leading to decompensation.
- Metabolic disturbances: Assuming that all altered mental status is due to intoxication only.

DISCLOSURE

The authors have no actual or potential conflicts of interest in relation to this article.

AUTHORSHIP

All authors had access to the data and played a role in writing this article.

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